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Compulsive exercise or exercise dependence? Clarifying conceptualizations of exercise in the context of eating disorder pathology

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Abstract

Objective—Maladaptive exercise relates to eating disorder (ED) pathology and impairment in clinical and non-clinical populations. At present, two different conceptualizations of maladaptive exercise are often studied in relation to ED pathology: compulsive exercise and exercise dependence. Compulsive exercise functions to avoid negative affect (e.g., guilt and anxiety) associated with not exercising, whereas exercise dependence is associated with tolerance to exercise benefits and avoidance of exercise withdrawal. At present, clinicians and researchers struggle to determine the most appropriate term for describing problematic exercise in individuals with ED pathology. This study aimed to directly compare these conceptualizations of maladaptive exercise in relation to severity of ED pathology.

Design—This study examined cross-sectional data.

Method—Undergraduate participants (N=235, 78% female) with elevated ED pathology completed the Eating Disorder Examination Questionnaire (EDE-Q), Compulsive Exercise Test (CET), and Exercise Dependence Scale (EDS). Multiple linear regression analyses evaluated associations between EDE-Q and CET and EDS scores and dominance analysis determined which qualities of exercise were uniquely associated with EDE-Q scores.

Results—Results suggest that compulsive qualities of exercise, including exercise to control shape and weight and to avoid negative affect are more strongly associated with severity of ED pathology than qualities of exercise dependence.

Conclusions—Clinicians and researchers working with ED populations can benefit from prioritizing assessments that capture compulsive qualities of exercise. Additionally, these results suggest that interventions that effectively target other compulsive behaviors (e.g., exposure and

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Declarations of interest

None.

response prevention) may be promising treatment options for problematic exercise in the context of EDs.

Keywords

Eating disorders; Maladaptive exercise; Compulsive exercise; Exercise dependence

1. Introduction

Eating disorders (EDs) are serious mental illnesses associated with significant medical, personal, familial, and societal risks (Schaumberg, et al., 2017) that can include maladaptive alterations in physical activity levels. As many as 80% of individuals with anorexia nervosa (AN) and 40% of individuals with bulimia nervosa (BN) report problematic exercise behaviors (Brewerton, Stellefson, Hibbs, Hodges, & Cochrane, 1995; Dalle Grave, Calugi, & Marchesini, 2008; Shroff et al., 2006). Although physical activity often supports features of positive mental health, the relation between exercise and EDs is complex and, at present, poorly understood. Further, maladaptive exercise in the context of EDs is associated with increased likelihood of suicidal behavior (Smith et al., 2013), worse treatment outcome (Dalle Grave et al., 2008), poor disorder prognosis (Carter, Blackmore, Sutandar-Pinnock, & Woodside, 2004), and lower quality of life (Cook et al., 2014a). Problematic exercise also clusters with several traits associated with increased psychopathology and impairment including: obsessive-compulsive traits and symptoms (Davis & Kaptein, 2006; Shroff et al., 2006); anxiety (Holtkamp, Hebebrand, & Herpertz-Dahlmann, 2004; Shroff et al., 2006); perfectionism (Shroff et al., 2006); body dissatisfaction (Brewerton et al., 1995); reward dependence (Dalle Grave et al., 2008); dietary restraint (Dalle Grave et al., 2008; Holtkamp, Hebebrand, & Herpertz-Dahlmann, 2004); anhedonia (Davis & Woodside, 2002); and hypoleptinemia (Holtkamp et al., 2003; Holtkamp et al., 2006). Curtailing maladaptive exercise in the treatment of EDs is difficult (Bratland-Sanda et al., 2010) and this symptom can both interfere with treatment progress and increase risk for relapse (Dalle Grave et al., 2008; Solenberger, 2001; Steinhausen et al., 2008, Grigoroiu-Serbanescu, Boyadjieva, Neumärker, & Metzke, 2008). Thus, it is important to effectively treat problematic exercise. Understanding factors contributing to the development and maintenance of maladaptive exercise can inform the development of effective treatments; however, at present, several divergent conceptualizations exist in the literature.

1.1. Development and maintenance of problematic exercise

Problematic exercise was included in the earliest descriptions of AN (Gull, 1874), and was initially termed “excessive”. Subsequently, descriptions of exercise behavior in the context of EDs focused primarily on frequency, intensity, and duration (Davis & Fox, 1993). More recent evidence suggests that an individual’s psychological relationship to exercise (e.g., the degree of distress associated with fluctuations in physical activity routine) is more informative than quantity of exercise in predicting negative outcomes, ED pathology (Adkins & Keel, 2005; Meyer & Taranis, 2011; Mond & Calogero, 2009; Seigel & Hetta, 2001), and recovery (Bardone-Cone et al., 2016). As such, the reinforcing qualities of exercise, including its anxiolytic (Favaro, Caregaro, Burlina, & Santonastaso, 2000; Guarda, Schreyer, Boersma, Tamashiro, & Moran, 2015) and naturally rewarding (Davis &

Woodside, 2002; Guarda et al., 2015) properties, may play a role in the development of maladaptive exercise behaviors. The activity-based anorexia model (ABA), an animal-based model of AN, demonstrates that rodents housed with running wheels and subjected to food restriction reduce their food intake and increase running wheel activity over time, to the point of starvation and death (Chowdhury, Chen, & Aoki, 2015). Consistent with this model, human studies of the course of illness suggest that elevations in activity often occur early in the course of AN (Achamrah, Coëffier, & Déchelotte, 2016; Davis et al., 1995).

Following the initiation of problematic exercise, psychological processes may explain the maintenance of maladaptive exercise patterns. Pathological exercise, independent of ED pathology, can be explained as obligatory (Thompson & Pasman, 1991) compulsive (Cook, Hausenblas, & Freimuth, 2014a,b; Dalle Grave et al., 2008; Dittmer, Jacobi, & Voderholzer, 2018; Meyer & Taranis, 2011), excessive (Long, Smith, Midgley, & Cassidy, 1993), overcommited (Yates, Shisslak, Crago, & Allender, 1994), addictive (Adams, 2002), and a dependence akin to substance dependence (Adams, 2009; Bamber, Cockerill, Rodgers, & Carroll, 2003; Bratland; Cook, Hausenblas, Tuccitto, & Giacobbi, 2011; Hausenblas & Downs, 2002; Sanda et al., 2011). Additionally, pathological exercise can be defined as either a *primary* disorder or a *secondary* symptom of an ED (Bamber, et al., 2003). Primary pathological exercise functions to reduce negative affect. In contrast, secondary pathological exercise is conceptualized as functioning to control body weight and shape. Research distinguishing these forms of problematic exercise typically qualifies primary pathological exercise as addictive and secondary pathological exercise as compulsive (Cunningham, Pearman, & Brewerton, 2016). However, recent research indicates that individuals with pathological exercise in conjunction with ED pathology experience greater levels of both the addictive and compulsive qualities of exercise than individuals without EDs (Cook et al., 2013; Cunningham et al., 2016).

Due to the diversity of terms used to describe problematic exercise, recent work has examined conceptual overlap between these terms and suggests that problematic exercise exists in two main forms (Cunningham et al., 2016): a behavioral addiction (Adams & Kirkby, 2002; Bamber et al., 2003; Bratland-Sanda et al., 2011; Cook et al., 2011) and/or a compulsive behavior (Cook, Hausenblas, & Freimuth, 2014b; Dalle Grave et al., 2008). At present, multi-dimensional formulations of compulsive exercise (Meyer, Taranis, Goodwin, & Haycraft, 2011) and exercise dependence (Hausenblas & Downs, 2002) provide comprehensive models with proposed mechanisms that contribute to the initiation and maintenance of pathological exercise among individuals with elevated ED pathology.

Compulsive exercise—Persistent and excessive behaviors (in this case, exercise), performed to relieve anxiety or distress associated with perceived negative consequences when abstaining from the behavior, are compulsive and do not necessarily lead to reward or pleasure (Chamberlain, Stochl, Redden, & Grant, 2018; Godier & Park, 2014; Robbins, Gillan, Smith, de Wit, & Ersche, 2012). In the context of EDs, compulsive exercise can function to avoid anxiety associated with the consequences of not exercising, including weight gain and body shape changes (Dittmer et al., 2018; Meyer et al., 2011; Meyer & Taranis, 2011). In addition to this negative reinforcement (i.e., avoidance of anxiety, shame, and guilt) that maintains physical activity, Meyer and colleagues' (2011) multi-dimensional

model of compulsive exercise posits that exercise behaviors are also maintained by positive reinforcement (i.e., mood improvement). Additionally, this model suggests that specific dispositional traits (e.g., perfectionism, rigidity) can enhance risk for compulsive exercise by influencing the frequency, intensity and regulation of negative emotions resulting from abstaining from exercise.

Exercise dependence—Alternatively, pathological exercise has been conceptualized as a cluster of cognitive, behavioral, and physiological symptoms, similar to those of substance dependence disorders (Adams & Kirkby, 2002; Allegre, Souville, Therme, & Griffiths, 2006; Bamber et al., 2003). Conceptualization of exercise dependence is modeled after the Diagnostic and Statistical Manual for Mental Disorder's (DSM-IV; American Psychiatric Association, 2000) definition of substance dependence (Bamber et al., 2003; Hausenblas & Downs, 2002) and is defined by three or more of the following presentations: 1) Tolerance, (i.e., the need for increased amounts of exercise to achieve desired effects or diminished effects with continued use of the same amount of exercise), 2) Withdrawal (aversive psychological and physiological symptoms associated with exercise cessation), 3) Intention Effects (i.e., exercising more or for longer than was intended), 4) Lack of Control (i.e., unsuccessful efforts to reduce exercise), 5) Time (i.e., large amounts time devoted to exercise), 6) Reduction in Other Activities (i.e., exercising rather than engaging in social, occupational, or recreational activities) and 7) Continuance (i.e., persistent exercise despite negative physical and/or psychological consequences; Hausenblas & Downs, 2002). This model likens exercise dependence to other forms of behavioral addiction (e.g., gambling, substance misuse), which are robustly supported by literature confirming the neurobiological reward pathways in the maintenance of these behavior patterns (Potenza, 2013).

1.2. Need for resolution of existing theories

At present, compulsive exercise and exercise dependence represent different theoretical models for the maintenance of exercise behaviors in EDs, whereby exercise behaviors are primarily reinforced by either reduction in negative affect or alternatively, by rewarding experiences (Bratland-Sanda et al., 2011; Cook et al., 2011; Cook et al., 2014b; Davis & Claridge, 1998; Davis & Kaptein, 2006; Dittmer et al., 2018; Guarda et al., 2015; Herring, Sailors, & Bray, 2014). Conceptualizing exercise as a compulsive behavior implies that exercise functions to reduce negative affect (e.g., anxiety). In contrast, a definition of exercise dependence contends that the rewarding properties of physical activity drive exercise behaviors.

As adequately assessing pathological exercise is difficult and inconsistent, delineating the extent to which psychological qualities of exercise (i.e., compulsive exercise versus exercise dependence) describe pathological exercise in the context of eating pathology will inform screening efforts for EDs. Additionally, clinicians who work with individuals diagnosed with EDs struggle with how best to treat individuals who exhibit heightened activity patterns (Noetel, et al., 2016). If exercise serves an anxiety-reducing function (i.e., compulsive exercise), interventions that enhance distress tolerance, and provide exposure to anxiety-inducing situations while preventing exercise as a coping strategy (i.e., exposure and response prevention) may be most effective. Alternatively, if exercise demonstrates

associations with reward-related motivation (i.e., exercise dependence), interventions that target reward-based learning (e.g., stimulus control) may be indicated as intervention strategies.

Both compulsive exercise and exercise dependence are associated with ED pathology. Although, at present, one conceptualization is not conclusively favored over the other, recent work suggests that compulsive exercise is highly correlated with ED pathology (Cunningham et al., 2016; Taranis, Touyz, & Meyer, 2011) and a majority of experts in the field of EDs report that compulsive exercise best describes pathological exercise among individuals with EDs (Noetel, Dawson, Hay, & Touyz, 2017). Additionally, among individuals with ED pathology, pathological exercise is associated with obsessive-compulsive disorder (OCD: Davis, 2006; Naylor, Mountford, & Brown, 2011; Noetel et al., 2016). Finally, recent genetic analyses indicate that polygenic scores indexing risk for both AN and OCD predict compulsive exercise in an epidemiological sample of adolescents (Yilmaz, Schaumberg, Crowley et al., 2019), providing further indication that the relationship between risk for compulsivity and maladaptive exercise in EDs may be biologically rooted. Together, this evidence demonstrates consistent support that compulsive qualities of exercise are relevant for those with ED pathology and that; similar to OCD symptoms, the negative affect reducing functions of compulsive exercise are particularly important.

1.3. Current study

The current study aimed to compare conceptualizations of maladaptive exercise in relation to ED pathology and to further identify whether specific facets of these conceptualizations drive associations with ED pathology. More specifically, we explored which qualities of pathological exercise were most strongly associated with ED pathology by directly comparing the extent to which the Compulsive Exercise Test (CET; Taranis et al., 2011) and Exercise Dependence Scale (EDS; Hausenblas & Downs, 2002) explained variation in global scores on the Eating Disorder Examination-Questionnaire (EDE-Q; Fairburn & Beglin, 1994). Consistent with recent work linking compulsive exercise to ED pathology we hypothesized that eating pathology would demonstrate a stronger association with compulsive qualities of exercise. Additionally, the associations between specific components of compulsive exercise and exercise dependence were examined in relation with EDE-Q scores. We anticipated that the negatively reinforcing qualities (i.e., avoidance of negative affect and weight control) of exercising would be most related ED pathology.

2. Methods

2.1. Participants and procedure

Undergraduate students (78.4% females; N=235) from a large northeastern university in the United States were recruited through introductory psychology classes and received course credit in exchange for study participation. Participants with ED pathology, as determined by scores on the EDE-Q with the above-established clinical cutoffs (Mond, Hay, Rodgers, Owen, & Beumont, 2004; Schaefer et al., 2018), were relatively young (M = 18.84, SD = 1.56) and self-identified as Caucasian (48.9%), Black or African American (18.3%), Asian

(14.9%), Hispanic or Latino (14%), or other (3.9%). Based on established criteria (Centers for Disease Control and Prevention, 2017), body mass indices (M = 26.22, SD =5.22) classified participants as underweight (2.1%), normal weight (41.3%), overweight (37.9%), and obese (14.5%). Ten participants (4.3%) did not provide height and weight data.

All participants attended a single laboratory appointment during which they completed informed consent, provided demographic information, and completed a series of questionnaires on an online survey platform. Questionnaires relevant to the current study are described below. At the end of the laboratory appointment, research assistants recorded height and weight data using standardized laboratory equipment. The Institutional Review Board at this university approved study protocols.

2.2. Measures

Eating Disorder Examination - Questionnaire (EDE-Q; Fairburn & Beglin, 1994)—The EDE-Q is a 36-item self-report questionnaire designed to measure cognitive and behavioral symptoms of EDs. Participants reported their experience of ED-related behaviors (e.g., purging) and cognitive symptoms (e.g., fear of weight gain) over the past 28 days using a 7-point Likert scale. Responses to relevant items can be averaged to compute four subscales reflecting dietary restraint, eating concern, body shape concern, and weight concern. Averaging scores across these subscales provides an index of overall ED pathology. This index, global EDE-Q score, was the primary measure of ED pathology in the current study. In clinical and non-clinical ED samples, the EDE-Q has demonstrated high internal consistency, test-retest reliability, and correlations with other measures of ED pathology (see Berg, Peterson, Frazier, & Crow, 2012 for review). In the current study, this scale demonstrated excellent internal consistency (Cronbach's α = 0.85).

Additionally, an established clinical cutoff global EDE-Q score was used to determine study eligibility; cutoff scores for study inclusion were 2.30 for female participants (Mond et al., 2004) and 1.68 for male participants (Schaefer et al., 2018). These cutoff scores have demonstrated concurrent validity with other measures that determine ED cases and non-cases (Mond et al., 2004; Schaefer et al., 2018).

Compulsive Exercise Test (CET; Taranis et al., 2011)—The CET is a 24-item self-report questionnaire designed to assess cognitive and behavioral components of compulsive exercise. This measure consists of five subscales reflecting specific compulsive qualities of exercise. The following are examples of items for each of the five subscales: *avoidance and rule-driven behavior* (e.g., I feel guilty if I miss an exercise session), *weight control exercise* (e.g., If I cannot exercise, I fear I will gain weight), *mood improvement* (e.g., I feel happier and/or more positive after I exercise), *lack of exercise enjoyment* (e.g., I find exercise a chore), and *exercise rigidity* (e.g., My weekly pattern of exercise is repetitive). Participants rated the degree to which each statement was true for them on a 6-point Likert scale ranging from 0 (*never true*) to 5 (*always true*). Subscale scores reflect the mean of relevant items and total score is sum of the five subscales. This CET has demonstrated high reliability and validity in previous studies (Taranis et al., 2011) and demonstrated high internal consistency in the present sample (Table 1).

Exercise Dependence Scale (EDS; Hausenblas, Downs, Uso, Per, & Ricerca, 2002)—The EDS is a 21-item self-report questionnaire that assesses physical and psychological symptoms of exercise dependence. Respondents rated each item using a 6-point Likert scale ranging from 1 (*never*) to 6 (*always*). This scale includes three items for each of the seven subscales Example items for each of the subscales include: *tolerance*: 'I continually increase my exercise frequency to achieve the desired effects/benefits', *withdrawal*: "I exercise to avoid feeling irritable", *intention effect*: "I exercise longer than I intend", *lack of control over exercise:* "I am unable to reduce how long I exercise", *time:* "I spend a lot of time exercising", *reduction in other activities:* "I would rather exercise than spend time with family/friends", *and continuance:* "I exercise when injured" Responses on the EDS can be used to classify individuals as exercise dependent, at risk for exercise dependency, or asymptomatic. Additionally, continuous subscale scores reflect sums of relevant items on the EDS and total EDS score is the mean of all items. Continuous total and subscale scores, in which higher scores reflect more severe exercise pathology, are valid and reliable measures exercise dependence (Downs, Hausenblas & Nigg, 2002; Monok et al., 2012), and demonstrated high internal consistency in the present sample (Table 1).

2.3. Analytic plan

Missing data were replaced with estimation-maximization procedures and univariate outliers were deleted. Bivariate correlations examined associations between EDE-Q scores and full and subscale measures of the CET and EDS. Due to the large number of comparisons (N =48), Bonferroni corrections were applied resulting in an adjusted significance level of p < .001. Next, multiple regression analysis, controlling for BMI and gender, examined the extent to which EDS and CET total scores predicted EDE-Q global scores. Hierarchical multiple linear regression analyses were conducted, in which covariates were entered in the first step and CET and EDS total scores in the second step. Follow-up dominance analysis determined the unique contributions of compulsive and addictive exercise to ED pathology. Dominance analysis is a statistical method used to determine the unique variance (R^2) in the dependent variable (EDE-Q) accounted for by each predictor in the model. This method accounts for multicollinearity of predictors by averaging a predictor variable's contribution to the dependent variable across all possible models containing individual predictor variables and all possible combinations of predictor variables (Budescu, 1993). Dominance analysis also facilitates pairwise comparisons of the effects of independent variables on the dependent variable by computing the independent variable effect sizes for all models including all possible combinations of predictors. 'Dominant' predictors consistently account for more variance in the dependent variable across all models. In the present study, dominance weights were computed using an excel macro created by Braun and Oswald (2011). Comparisons of effect sizes from the final model determined whether CET or EDS total scores were most related to ED pathology. Then effect sizes for each predictor variable were compared across all models to determine dominance. Based on the results from these analyses, a similar method was used to further examine which specific subscales of the dominant scale (EDS or CET) were most strongly associated with ED pathology.

3. Results

3.1. Bivariate correlations

Descriptive statistics and bivariate correlation coefficients between variables of interest appear in Table 1. As expected, CET total score was positively correlated with EDE-Q global score. Additionally, the avoidance of negative affect and weight control subscales of the CET were positively correlated with ED pathology. The total EDS score was not significantly associated with EDE-Q global score; however, the withdrawal subscale was positively associated with ED pathology. With the exception of the lack of exercise enjoyment subscale of the CET, all subscale scores of the CET and EDS were positively associated with total scores of the CET and the EDS. The lack of exercise enjoyment subscale of the CET was not correlated with total CET score and was negatively associated with total EDS score.

3.2. Associations between CET, EDS, and EDE-Q scores

Hierarchical regression analyses, including EDS and CET total scores, yielded a significant final model ($F(4,218) = 14.68$, $p < .001$, adjusted $R^2 = 0.20$) that explained significantly more variance in EDE-Q scores than the model including only the covariates ($\Delta R^2 = 0.17$, $F(2, 214) = 22.82$, $p < .001$). Standardized and unstandardized regression coefficients and confidence intervals appear in Table 2. BMI and gender were significant covariates in this model and greater total CET scores were significantly associated with elevated EDE-Q global scores. EDS scores were negatively associated with EDE-Q global scores when included in the model with CET and covariates. Across models, CET total score dominated (all dominance R^2 values > 0.13) other predictor variables (all dominance R^2 values < 0.06) indicating that CET total score accounted for more variance in EDE-Q global score than BMI, gender, and EDS. Dominance R^2 values from the model including all predictors are presented in Table 2.

3.3. Associations between CET subscales and EDE-Q global scores

Because CET total scores dominated EDS total score in the initial regression model predicting EDE-Q global scores, subsequent multiple regression analyses determined the quality of compulsive exercise most strongly associated with ED pathology. The model including each of the CET subscales ($F(7, 211) = 13.86$, $p < .001$, adjusted $R^2 = 0.30$) explained a greater amount of variance in EDE-Q score ($\Delta R^2 = 0.27$, $F(5, 211) = 16.45$, $p < .001$) as compared with the model including only covariates (i.e., gender and BMI). Regression coefficients and dominance R^2 values appear in Table 3. Because the inclusion of covariates would have necessitated presentation of 127 unique submodels, they were not included in follow-up dominance analyses. Exercising for weight control dominated (all dominance R^2 values > 0.13) all other predictor variables (all dominance R^2 values < 0.11) across submodels in which it was included as a predictor variable. Notably, exercising to avoid negative affect was also a dominant predictor (all dominance R^2 values > 0.08), accounting for more variance in ED pathology than all predictors (all dominance R^2 values < 0.04), except weight control exercise, across models.

4. Discussion

Our results extend the current understanding of exercise within the context of eating pathology by directly comparing associations between two dominant conceptualizations of problematic exercise: compulsive exercise and exercise dependence and ED pathology. In support of hypotheses, compulsive qualities of exercise accounted for a greater proportion of variance in ED pathology than exercise dependence. This finding that, when directly compared to exercise dependence, compulsive exercise is more strongly related to ED pathology extends recent findings supporting associations between compulsive qualities of exercise and ED pathology (Cunningham et al., 2016; Taranis et al., 2011) and supports prior work linking EDs to compulsive qualities of exercise (e.g., Noetel et al., 2016; Yilmaz et al., 2019). Further, consistent with our hypotheses and previous research (e.g., Naylor et al., 2011; Taranis et al., 2011; Yilmaz et al., 2019), specific facets of compulsive exercise, (i.e., exercising to avoid negative affect and to control weight) were most strongly associated with elevated ED pathology.

Findings from this study assist in reconciling confusion about the most appropriate definition of problematic exercise in relation to ED pathology, enabling more efficient and consistent assessment. Specifically, our results suggest that researchers evaluating exerciserelated ED risk should prioritize measures that capture compulsive exercise, such as the CET (Taranis et al., 2011) or a recently developed clinical interview for compulsive exercise (Dittmer et al., 2018), over measures that focus primarily on exercise dependence. Further, we recommend that researchers with length limitations on assessment protocols consider including measures that specifically assess exercising for weight control and avoidance of negative affect.

These results also cast doubt on the notion that exercise in the context of eating pathology is driven primarily by reward-seeking. Although some theoretical models posit that driven exercise and other compulsive behaviors may initially develop via appetitive pathways that evolve into aversive control over time (e.g. avoidance of withdrawal; Bratland-Sanda et al., 2011; Cook et al., 2011; Davis & Claridge, 1998; Hausenblas & Downs, 2002), evidence from the current study indicates that reward-related measures were only slightly associated with eating pathology in this sample, suggesting that the role of *explicit* appetitive processes (i.e. those which are captured by self-report) in pathological exercise may be limited.

As an immediate next step, results from this investigation should be replicated in clinical populations of individuals with more severe EDs, along with following at-risk samples prospectively to elucidate the development of compulsive exercise and the transition from healthy to maladaptive exercise. Additionally, there may be variation in the nature of pathological exercise for those with different ED diagnoses (e.g., anorexia nervosa; bulimia nervosa), therefore, future research can compare conceptualizations of problematic exercise within diagnostic classes. Future research might also include examination of other compulsive and addictive behaviors and traits (e.g., perfectionism, rewardresponsivity) and the impacts of these traits on manifestations of maladaptive exercise. This work will allow for improved identification of risk for either compulsive dependent exercise behaviors. If results from the current investigation are replicated in other samples, considering effective

interventions for compulsive behaviors (e.g., exposure and response prevention) may be promising approaches to treating pathological exercise among individuals with comorbid eating pathology.

There are several strengths of the current study including examination of a large sample of individuals with elevated ED pathology, which allowed for adequate power to examine moderate effects and dominance of these measures. Additionally, this study represents the first investigation to include direct comparison between established measures of pathological exercise using novel statistical methodology. Finally, college students represent a vulnerable group for the development and escalation of EDs (Eisenberg, Nicklett, Roeder, & Kirz, 2011), thus our sample of college-aged individuals reporting elevated levels of eating pathology represents an appropriate population for this study. Despite these strengths, this study is limited in its reliance on retrospective self-reports of compulsive exercise and exercise dependence. Further, the measurement instruments included in this study may impact interpretation of our results. Although the CET is the leading instrument for assessing compulsive exercise, this instrument was developed specifically to examine associations between exercise and ED pathology (Taranis et al., 2011). In contrast, although the EDS has been commonly applied to samples with ED pathology (e.g., Cook et al., 2011) this measure was originally developed to assess exercise pathology independent of ED pathology (Hausenblas & Downs, 2002). Additionally, our inclusion of the EDS in this study allowed for assessment of exercise dependence, which may differ from other conceptualizations of pathological exercise (i.e., exercise addiction); thus, the results from this study cannot definitively establish compulsive exercise as the dominant conceptualization of problematic exercise. Future research utilizing alternative measurement methodologies (e.g., clinical interviews, ecological momentary assessment) that capture nuanced characteristics of pathological exercise and affective and cognitive experiences before and after exercise will further elucidate the nature of problematic exercise among those with elevated ED pathology. This study also relied on cross-sectional data. Additional studies that examine associations between ED pathology and compulsive and addictive exercise longitudinally and at earlier developmental time points will improve our understanding of the extent to which findings in the current study generalize to the broader context of ED risk.

5. Conclusions

This study provides clarification about the nature of maladaptive exercise among individuals with EDs. Overall, these findings suggest that compulsive qualities of exercise, including the alleviation of negative affect and/or reduction in discomfort associated with possible changes in weight or shape, are more relevant to ED pathology than the rewarding effects of physical activity associated with exercise dependence. These results contrast traditional conceptualizations that maladaptive exercise initially develops as a positively reinforcing behavior and transitions to aversive control over time and emphasize the importance of the negative reinforcing aspects of exercise for individuals with EDs. Researchers and clinicians can benefit from the findings of this study by attending to the affective regulation and weight control functions of exercise when assessing and treating problematic exercise among individuals with EDs. In so doing, researchers can more precisely focus on relevant

maladaptive exercise among individuals with EDs. Finally, by focusing on the compulsive components of exercise, clinicians working with EDs can implement targeted and effective interventions.

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Table 1

Descriptive statistics, reliability coefficients and bivariate correlations for variables of interest.

Variable	*M*	*SD*	Cronbach's α	EDE-Q	CET	EDS
EDE-Q Global	2.85	.88	.85	–	.37[a]	.09
EDS Total	52.09	19.16	.94	.09	.58[a]	–
Withdrawal	8.77	4.13	.88	.19[a]	.62[a]	.80[a]
Continuance	7.19	3.82	.85	.11	.40[a]	.69[a]
Tolerance	10.28	4.58	.93	.00	.54[a]	.81[a]
Lack of Enjoyment	6.15	3.12	.85	.08	.34[a]	.66[a]
Reduction	5.39	2.44	.64	.09	.41[a]	.69[a]
Time	7.14	3.41	.89	.00	.47[a]	.84[a]
Intention	7.17	3.69	.94	.03	.35[a]	.79[a]
CET Total	13.29	3.36	.88	.37[a]	–	.58[a]
Avoidance of Negative Affect	1.85	1.21	.91	.32[a]	.78[a]	.68[a]
Weight Control	3.60	0.97	.71	.41[a]	.69[a]	.38[a]
Mood Improvement	3.12	1.27	.85	.13	.79[a]	.56[a]
Lack of Exercise Enjoyment	2.00	1.23	.77	.06	−.27	−.65[a]
Rigidity	2.72	1.36	.80	.16	.81[a]	.67[a]

Note. Gender was dichotomously coded 0=male, 1=female; BMI= Body Mass Index; CET= Compulsive Exercise Test; EDE-Q=Eating Disorder Examination-Questionnaire; EDS=Exercise Dependence Scale; Italics indicate subscales of the EDS and CET.

[a] $p < .001$ (modified statistical significance accounting for 48 comparisons)

Table 2

Coefficients and dominance weight for regression model predicting EDE-Q from EDS and CET.

Predictor	*b*	SE (*b*)	*β*	*t*	95% CI	Dominance R^2
BMI	.02	.01	.09	1.44	−.01, .04	.01
Gender	**.51**	**.13**	**.25**	**3.96**	**.26, .76**	.05
EDS Total	−.01	.00	−.14	−1.79	−.01 .00	.01
CET Total	**.12**	**.02**	**.47**	**6.36**	**.09, .16**	.14

Note. BMI=body mass index, EDE-Q=eating disorder examination – questionnaire, EDS=exercise dependence scale, CET – compulsive exercise test. Bold values indicate significance at ($p < .05$).

Table 3

Coefficients and dominance weights for regression model predicting EDE-Q from CET subscales.

Predictor	*b*	SE (*b*)	*β*	*t*	95% CI	Dominance R^2
BMI	.01	.01	.08	1.31	−.01, .03	–
Gender	**.39**	**.13**	**.19**	**3.09**	**.14, .63**	-
AV	**.29**	**.06**	**.40**	**4.94**	**.17, .40**	**.09**
WC	**.34**	**.06**	**.36**	**5.32**	**.21, .46**	**.13**
MI	−.07	.06	−.11	−1.32	−.18, .04	.01
LE	**.19**	**.05**	**.27**	**3.85**	**.09, .29**	**.04**
RG	−.01	.06	−.02	−.22	−.12, .10	.01

Note. AV=avoidance of negative affect; WC=weight control; MI=mood improvement; LE=lack of exercise enjoyment; RG=rigidity. Bold values indicate significance at ($p < .05$). Italics indicate subscales of the CET.